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BRADLEY ARANT ROSE & WHITE, LLP			AEDER, SEAN E	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/813,977	DYNAN ET AL.	
	Examiner	Art Unit	
	Sean E. Aeder	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --
Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) Responsive to communication(s) filed on 17 September 2007.
- 2a) This action is **FINAL**. 2b) This action is non-final.
- 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) Claim(s) 1-6, 8, 9, 11-13, 15-17, 19, 27-32 and 34-39 is/are pending in the application.
 - 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) Claim(s) 6, 8, 9, 11-13, 15-17, 19 and 27-31 is/are allowed.
- 6) Claim(s) 1, 2, 4, 5, 32, 34-39 is/are rejected.
- 7) Claim(s) 3 is/are objected to.
- 8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) The specification is objected to by the Examiner.
- 10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner.
 Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
 Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
 - a) All b) Some * c) None of:
 1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) Notice of References Cited (PTO-892)
- 2) Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) Information Disclosure Statement(s) (PTO/SB/08)
 Paper No(s)/Mail Date _____.
- 4) Interview Summary (PTO-413)
 Paper No(s)/Mail Date. _____.
- 5) Notice of Informal Patent Application
- 6) Other: _____.

Detailed Action

The Amendments and Remarks filed 9/17/07 in response to the Office Action of 5/17/07 are acknowledged and have been entered.

Claims 1-6, 8, 9, 11-13, 15-17, 19, 27-32, and 34-39 are pending.

Claims 6, 8, 9, 11-13, 15-17, 19, 31, and 32 have been amended by Applicant.

Claims 1-6, 8, 9, 11-13, 15-17, 19, 27-32, and 34-39 are currently under examination.

Rejections Withdrawn

The rejection under 35 U.S.C. 112, first paragraph, is withdrawn in view of amendments.

Response to Arguments

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless —

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 2, 4, and 5 remain rejected under 35 U.S.C. 102(b), as being anticipated by Carter et al (Mol. and Cell. Biol., 1990, 10(12):6460-6471), as evidenced by Li et al (Nucleic Acids Research, 2003, 31(20):5848-5857) and the specification, is

maintained for the reasons stated in the Office Action of 5/17/07 and for the reasons set-forth below.

Claim 1 is drawn to a composition comprising a DNA repair modulator that specifically binds to SEQ ID NO:16 and inhibits non-homologous end joining. Claim 2 is drawn to the composition of claim 1 wherein the DNA repair modulator comprises a polypeptide. Claim 4 is drawn to the composition of claim 1, wherein SEQ ID NO:16 is located on a DNA-PKcs and said DNA repair modulator inhibits less than 50% of DNA-PKcs enzymatic activity. Claim 5 is drawn to a single chain antibody that specifically binds to DNA-PKcs in a region outside of the catalytic domain, wherein the single chain antibody comprises the CDRs SEQ ID NOs 18-23 in an immunological framework.

Carter et al teaches a monoclonal antibody, mAb 18-2, which was used in the production of a single chain antibody of Li et al and described in the specification (see left column of page 5849 of Li et al and page 38 of the instant specification, in particular). It is noted that said monoclonal antibody is equivalent to a single chain antibody in an immunological framework (see claim 5) and the Li et al reference is not used in this rejection to provide motivation to make anything. As evidenced by the specification (page 19 lines 30-31 and page 18 lines 24-29, in particular), the antibody of Li et al (generated from parental antibody mAb18-2, taught by Carter et al) comprises the CDRs encoded by instant SEQ ID NOs:18-23. Further, because the antibody of Li et al was generated from the antibody taught by Carter et al, the antibody taught by Carter et al *inherently* has the same CDRs, in an immunoglobulin framework, as the single chain antibody of Li et al and the specification (CDRs encoded by instant SEQ ID

NOs:18-23). Further, since the single chain antibody of Li et al binds SEQ ID NO:16 (see page 43 lines 12-15 and page 39 lines 17-22 of the specification, in particular), mAb 18-2 and the single chain antibody of Li et al have identical CDRs, and Western blot analysis of mAb 18-2 and the single chain antibody of Li et al demonstrate binding to the same region of DNA-PKcs (as evidenced by Figure 1C of Li et al), the antibody taught by Carter et al would bind SEQ ID NO:16, which is found on regions outside of the catalytic domain of DNA-PKcs. Further, as evidenced by Li et al (page 5850 right column, in particular), mAb 18-25 inhibits less than about 50% of DNA-PKcs enzymatic activity. The office does not have the facilities and resources to provide the factual evidence needed in order to establish that the product of the prior art does not perform the same function as the claimed product. In the absence of evidence to the contrary, the burden is on Applicant to prove that this function of the claimed product is different from that taught by the prior art and to establish patentable differences. See *In re Best* 562F.2d 1252, 195 USPQ 430 (CCPA 1977) and *Ex parte Gray* 10 USPQ 2nd 1992 (PTO Bd. Pat. App. & Int. 1989).

In the Reply of 9/17/07, Applicant argues that the ability of a single chain antibody to inhibit non-homologous end joining is not anticipated by the Carter reference. Applicant further argues that a single chain antibody of the present disclosure and mAb 18-2 display different binding characteristics. Applicant further argues that it has not been shown that the binding regions of the antibody taught by Carter et al and a single chain antibody of the present invention bind identical sequences. Applicant further argues that Figure 1C of Li and the present disclosure

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show different binding patterns in immunoblot experiments and concludes that the antibody taught by Carter et al and a single chain antibody of Li and the present disclosure show empirical differences in binding to DNA-PKcs under the same experimental conditions. Applicant further argues that the binding characteristics of a single chain antibody of the present disclosure could not be predicted from Carter et al. Applicant further argues that the activities displayed by a single chain antibody of the present disclosure and mAb 18-2 are unrelated to unexpected inhibition of non-homologous end joining. Applicant further argues that the Li reference, work of the inventors, is not proper in the present invention.

The amendments to the claims and the arguments found in the Reply of 9/17/07 have been carefully considered, but are not deemed persuasive. In regards to the argument that the ability of a single chain antibody to inhibit non-homologous end joining is not anticipated by the Carter reference, it is first noted that claims drawn to a single chain antibody comprising CDRs in an immunological framework are equivalent to mAbs comprising said CDRs. Further, as evidenced by Li et al, the antibodies taught by Carter et al inherently inhibit non-homologous end joining (see Figure 2 of Li et al, in particular). It is noted that the ability of the antibodies taught by Carter et al to inhibit non-homologous end joining is not a matter of impermissible hindsight, but rather a showing of an inherent property of the antibodies. The discovery of a previously unappreciated property of a prior art composition, or of a scientific explanation of the prior art's function, does not render the old composition patentably new to the discoverer. *Atlas Powder Co. v. Ireco Inc.*, 190 F. 3.d 1324, 1374, 51 USPQ2d 1943,

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1947 (Fed. Cir. 1999). Thus, the claiming of the unknown property which is inherently present in the prior art does not necessarily make the claim patentable.

In regards to the argument that a single chain antibody of the present disclosure and mAb 18-2 display different binding characteristics, said argument is not in commensurate with the scope of the claims. The rejected claims are not drawn to a single particular single chain antibody. Rather, the rejected claims are drawn to DNA repair modulators that bind particular sequences and single chain antibodies comprising particular CDRs in an immunoglobulin framework. For the reasons state above, mAb 18-2 inherently has the binding characteristics recited in the instant claims.

In regards to the argument that it has not been shown that the binding regions of the antibody taught by Carter et al and a single chain antibody of the present invention bind identical sequences, said argument is not in commensurate with the scope of the claims. The rejected claims are not drawn to a single particular single chain antibody. Rather, the rejected claims are drawn to DNA repair modulators that bind particular sequences and single chain antibodies comprising particular CDRs in an immunoglobulin framework. It is further noted that because the single chain antibody of Li et al binds SEQ ID NO:16 (see page 43 lines 12-15 and page 39 lines 17-22 of the specification, in particular), mAb 18-2 taught by Carter et al and the single chain antibody of Li et al have identical CDRs, and Western blot analysis of mAb 18-2 and the single chain antibody of Li et al demonstrate binding to the same regions of DNA-PKcs (as evidenced by Figure 1C of Li et al), the antibody taught by Carter et al would bind SEQ ID NO:16, which is found on regions outside of the catalytic domain of DNA-PKcs.

In regards to the argument that Figure 1C of Li and the present disclosure show different binding patterns in immunoblot experiments and concludes that the antibody taught by Carter et al and a single chain antibody of Li and the present disclosure show empirical differences in binding to DNA-PKcs under the same experimental conditions, said argument is not in commensurate with the scope of the claims. The rejected claims are not drawn to a single particular single chain antibody. Rather, the rejected claims are drawn to DNA repair modulators that bind particular sequences and single chain antibodies comprising particular CDRs in an immunoglobulin framework. It is further noted that the antibody taught by Carter et al *clearly* binds the same N-terminal region as the single chain antibody taught by Li et al (see Figure 1C).

In regards to the argument that the binding characteristics of a single chain antibody of the present disclosure could not be predicted from Carter et al, the binding characteristics of the antibody taught by Carter et al are inherent characteristics. As noted above, the discovery of a previously unappreciated property of a prior art composition, or of a scientific explanation of the prior art's function, does not render the old composition patentably new to the discoverer. *Atlas Powder Co. v. Ireco Inc.*, 190 F. 3.d 1324, 1374, 51 USPQ2d 1943, 1947 (Fed. Cir. 1999). Thus, the claiming of the unknown property which is inherently present in the prior art does not necessarily make the claim patentable.

In regards to the argument that the activities displayed by a single chain antibody of the present disclosure and mAb 18-2 are unrelated to unexpected inhibition of non-homologous end joining, characteristics of the antibody taught by Carter et al are

inherent characteristics. As noted above, the discovery of a previously unappreciated property of a prior art composition, or of a scientific explanation of the prior art's function, does not render the old composition patentably new to the discoverer. *Atlas Powder Co. v. Ireco Inc.*, 190 F. 3d 1324, 1374, 51 USPQ2d 1943, 1947 (Fed. Cir. 1999). Thus, the claiming of the unknown property which is inherently present in the prior art does not necessarily make the claim patentable.

In regards to the argument that Li et al is not proper in the present invention, Li et al is found in this rejection to *evidence inherent properties* of a product taught in the prior art. The teachings of Li et al are not used in this rejection as prior art; rather, Li et al is used to describe what others (Carter et al) have taught in the prior art.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

The rejection of claims 1, 2, 4, 5, 32, and 34-37 under 35 U.S.C. 103(a), as being unpatentable over Carter et al (Mol. and Cell. Biol., 1990, 10(12):6460-6471) as applied to claims 1, 2, 4, and 5 above, and further in view of Bejcek et al (Cancer Research, 1995, 55:2346-2351) and Schwarze et al (Science 9/3/99, 285:1569-1572), is maintained for the reasons stated in the Office Action of 5/17/07 and for the reasons set-forth below.

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Anticipation of claims 1, 2, 4, and 5 by Carter et al is described above. Carter et al does not specifically teach a single chain antibody comprising a protein transduction domain wherein the single chain antibody inhibits DNA repair by binding to a repair polypeptide and includes the CDR regions SEQ ID NOs:18-23 in an immunoglobulin framework. (claim 32), wherein the DNA repair polypeptide comprises DNA-PKcs (claim 34), wherein the single chain antibody binds to a region including SEQ ID NO:16 or a portion thereof (claims 35-36) and inhibits non-homologous end joining (claim 37). However, these deficiencies are made up in the teachings of Bejcek et al and Schwarze et al.

Bejcek et al teaches single chain antibodies constructed from mAbs (pages 2346-2347, in particular). Bejcek et al further teaches single chain antibodies overcome several problems associated with intact mAbs, particularly because of the large size of the mAbs and the resultant relative inability to penetrate tissue (page 2350, in particular).

Schwarze et al teaches delivery of proteins into cells by adding a protein transduction domain to said proteins (right column of page 1570, in particular).

One of ordinary skill in the art at the time the invention was made would have been motivated to produce a single chain antibody comprising the CDRs of the antibody taught by Carter et al (CDR regions SEQ ID NOs:18-23) in an immunoglobulin framework attached to a protein transduction domain because the target of the antibody taught by Carter et al is inside cells (see pages 642-643 of Carter et al) and Bejcek et al teaches single chain antibodies constructed from mAbs penetrate tissue better than

intact mAbs (page 2350, in particular) and Schwarze et al teaches adding a protein transduction domain to proteins enhances said protein's ability to enter cells (right column of page 1570, in particular). One of ordinary skill in the art at the time the invention was made would have had a reasonable expectation of success for producing a single chain antibody comprising the CDRs of the antibody taught by Carter et al (CDR regions SEQ ID NOS:18-23) in an immunoglobulin framework attached to a protein transduction domain because Bejcek et al teaches single chain antibodies constructed from mAbs (pages 2346-2347, in particular) and Schwarze et al teaches adding a protein transduction domain to said protein (right column of page 1570, in particular). Therefore, the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, absent unexpected results. Further, since the single chain antibody taught by Li et al binds SEQ ID NO:16 (see page 43 lines 12-15 and page 39 lines 17-22 of the specification, in particular), mAb 18-2 and the single chain antibody taught by Li et al have identical CDRs, and Western blot analysis of mAb 18-2 and the single chain antibody taught by Li et al demonstrate identical binding to DNA-PKcs (as evidenced by Figure 1C of Li et al), the single chain antibody taught by the combined teachings of Carter et al, Bejcek et al, and Schwarze et al would bind SEQ ID NO:16, which is found on regions outside of the catalytic domain of DNA-PKcs. Further, as evidenced by Li et al (page 5850 right column, in particular), mAb 18-2 inhibits less than about 50% of DNA-PKcs enzymatic activity. The office does not have the facilities and resources to provide the factual evidence needed in order to establish that the product of the prior art does not perform

the same function as the claimed product. In the absence of evidence to the contrary, the burden is on Applicant to prove that this function of the claimed product is different from that taught by the prior art and to establish patentable differences. See *In re Best* 562F.2d 1252, 195 USPQ 430 (CCPA 1977) and *Ex parte Gray* 10 USPQ 2nd 1992 (PTO Bd. Pat. App. & Int. 1989).

In the Reply of 9/17/07, Applicant cites the second paragraph on page 2346 and the discussion second of Bejcek and states having possession of a monoclonal antibody does not translate into having a single chain antibody that binds the same target and performs the same function as the original monoclonal antibody. Applicant further states that the failure rate of converting parental monoclonal antibodies into corresponding single chain antibodies was greater than 33% in the experiments of Bejcek. Applicant further cites Lamberski et al (Protein Expression and Purification, 47 pp82-92, 2006) and states that the single chain antibody taught by Lamberski et al displayed a different affinity to its target antigen as compared to a parental antibody. Applicant further cites Bose et al (Molecular Immunology, 40, pp617-31, 2003) and indicates that the single chain antibody taught by Bose et al displayed a different affinity to its target antigen as compared to a parental antibody. Applicant concludes that the assumption that a single chain antibody and the parent monoclonal antibody would inherently have the same binding characteristics and the same function is not supported by the references cited by the Examiner, Lamberski et al, or Bose et al.

The amendments to the claims and the arguments found in the Reply of 9/17/07 have been carefully considered, but are not deemed persuasive. In regards to the

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argument that Bejcek and states having possession of a monoclonal antibody does not translate into having a single chain antibody that binds the same target and performs the same function as the original monoclonal antibody, Bejcek et al clearly teaches methods *routinely* used in the art for developing single chain antibodies from parental monoclonal antibodies and screening for those that have functions of said parental monoclonal antibodies (right column of page 2349, in particular). Further, in regards to the argument that the failure rate of converting parental monoclonal antibodies into corresponding single chain antibodies was greater than 33%, the experiments of Bejcek et al clearly teach methods *routinely* used in the art for developing single chain antibodies from parental monoclonal antibodies and screening for those that have functions of said parental monoclonal antibodies wherein said functions were found in the majority of single chain antibodies tested (right column of page 2349 and Figure 5, in particular). Therefore, the method taught by Bejcek et al demonstrates that properly folded single chain antibodies of parental monoclonal antibodies are routinely produced and the single chain antibodies predictably bind the same target as the parental monoclonal antibodies.

In regards to the arguments based on the affinities of single chain antibodies and the teachings of Lamberski et al and Bose et al, Applicant is arguing limitations not recited in the claims. It is noted that the instant claims do not recite limitations based on particular affinities of the claimed products.

Claim Rejections - 35 USC § 103

Claims 1, 2, 4, 5, 32, and 34-39 remain rejected under 35 U.S.C. 103(a), as being unpatentable over Carter et al (Mol. and Cell. Biol., 1990, 10(12):6460-6471) in view of Bejcek et al (Cancer Research, 1995, 55:2346-2351) and Schwarze et al (Science 9/3/99, 285:1569-1572) as applied to claims 1, 2, 4, 5, 32, and 34-37 above, and further in view of Kelley et al (US Patent 6,252,048 B1; 6/26/01) or Jang et al (Molecular Breeding, 2002, 9:81-91), for the reasons stated in the Office Action of 5/17/07 and for the reasons set-forth below..

Anticipation of claims 1, 2, 4, 5, 32, and 34-37 by the combined teachings of Carter et al, Bejcek et al, and Schwarze et al is described above.

The combined teachings of Carter et al, Bejcek et al, and Schwarze et al does not specifically teach single chain antibodies comprising nuclear localization signals or chloroplast localization signals. However, these deficiencies are made up in the teachings of Kelley et al and Jang et al.

Kelley et al teaches a nuclear localization signal that was recombinantly added to a DNA repair protein to improve the nuclear localization of the protein (column 62 lines 25-28, in particular).

Jang et al teaches a protein comprised of a chloroplast localization signal (pages 82-83, in particular). Jang et al further teaches that adding a chloroplast localization signal to polynucleotide constructs expressed in plants enhances expression of the protein product (page 87-88 and Figure 5, in particular).

One of ordinary skill in the art at the time the invention was made would have been motivated to add a nuclear localization signal to the single chain antibody taught by the combined teachings of Carter et al, Bejcek et al, and Schwarze et al because Kelley et al teaches nuclear localization signals send protein constructs to the nucleus (column 62 lines 25-28, in particular) and Carter et al teaches the target of the single chain antibody is found in the nucleus (see abstract, in particular). One of ordinary skill in the art at the time the invention was made would have had a reasonable expectation of success for adding a nuclear localization signal to the single chain antibody taught by the combined teachings of Carter et al, Bejcek et al, and Schwarze et al because Kelley et al teaches adding nuclear localization signals to protein constructs (column 62 lines 25-28, in particular).

Further, one of ordinary skill in the art at the time the invention was made would have been motivated to add a chloroplast localization signal to the single chain antibody taught by the combined teachings of Carter et al, Bejcek et al, and Schwarze et al because Jang et al further teaches that adding a chloroplast localization signal to polynucleotide constructs expressed in plants enhances expression of the protein product (page 87-88 and Figure 5, in particular) and one of skill in the art would recognize that enhanced production of the single chain antibody would enable one of skill to perform more assays with said antibody. One of ordinary skill in the art at the time the invention was made would have had a reasonable expectation of success for adding a chloroplast localization signal to the single chain antibody taught by the combined teachings of Carter et al, Bejcek et al, and Schwarze et al because Jang et al

teaches adding chloroplast localization signals to protein constructs (pages 82-83, in particular). Therefore, the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, absent unexpected results.

In the Reply of 9/17/07, Applicant repeats arguments discussed above and argues that Kelley and Jang do not suggest alleged shortcomings of Carter.

The amendments to the claims and the arguments found in the Reply of 9/17/07 have been carefully considered, but are not deemed persuasive. Arguments based on this rejection are discussed above.

Allowable Subject Matter

Claims 6, 8, 9, 11-13, 15-17, 19, and 27-31 are allowed.

Claim 3 is objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including all of the limitations of the base claim and any intervening claims.

Conclusion

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 C.F.R. ' 1.136(a). A shortened statutory period for response to this Final Action is set to expire three months from the date of this action. In the event a first response is filed within two months of the mailing date of this Final Action and the advisory action is not mailed until after the end of the three-month shortened statutory

period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 C.F.R. 1.136(a) will be calculated from the mailing date of the advisory action. In no event will the statutory period for response expire later than six months from the date of this Final Action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sean E. Aeder, Ph.D. whose telephone number is 571-272-8787. The examiner can normally be reached on M-F: 8:30-5:00.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Shanon Foley can be reached on 571-272-0898. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

SEA

/Misook Yu/
Primary Examiner
Art Unit 1642